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=> s IgE fusion
L2 21 IGE FUSION

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L3 ANSWER 1 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
2004:718668 Document No. 141:237093 Development of a ligand screening method
using wild type and mutant receptor proteins that enables segregation of
agonist and antagonist. Matsui, Kazuhiro; Ishibashi, Takuya; Oka,
Masanori (Toyo Boseki Kabushiki Kaisha, Japan). PCT Int. Appl. WO
2004074473 A1 20040902, 61 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT,
AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM,
DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE,
KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ,
NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR,
TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD,
RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI,
FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR.
(Japanese). CODEN: PIXXD2. APPLICATION: WO 2003-JP822 20030129.

AB A ligand screening method using wild type and mutant receptor proteins
that enables segregation of agonist and antagonist has been developed.
The mutant receptor proteins has amino acid alternations lowering binding
affinity for agonist or antagonist and the amino acid alternations include
conversion to amino acids with side chains of different size and the same
charge or conversion to side chains with different charges. Altered amino
acid can be the one that is not involved in binding to known agonists but
in binding to known antagonists. The used receptors can be selected among
nuclear receptors, membrane receptors, transport proteins, cofactors,
co-activators and polypeptides. More specifically, the receptor can be
selected among receptors for androgen, estrogen, glucocorticoid,
mineralocorticoid, progesterone, pregnane, vitamin D, thyroid hormones,
retinoic acid, retinoid, AHR (aryl hydrocarbon receptor), and peroxisome
proliferator-activated receptors from natural or recombinant animal,
plant, microbial resources. The used receptor mols. are fusion or
conjugates with proteins, carbohydrates, polymers, phosphoric acid or
sulfonic acid. The fusion partners can be selected among sugar-binding
proteins, glutathione-binding proteins, calcium-binding proteins,
poly-histidine, biotin-binding proteins, Ig-binding proteins, Igs (G, M,
A, E or D) and their fragments or fluorogenic conjugates. Human estrogen
receptor α and its variants (Asp at position 351 converted to Asn,
Gly, Glu or Ser) fusion protein with sugar-binding protein are
specifically used for screening of SERMs (Selective Estrogen Receptor
Modulators). Ligand screening process includes the judgement of the

ligand classification to agonists or antagonists based on the determined binding affinity to wild type or variant receptors using IC50(variant)/IC50(wild type) value as a parameter. A estrogen receptor α and variant fusion protein with mannose-binding protein were produced and the reliability of the binding affinity assay using the fusion receptor proteins was demonstrated by using various known ligands (17 β -estradiol, hydroxytamoxifen, tamoxifen, diethylstilbestrol and nonylphenol) with tritium-labeled 17 β -estradiol reference. The method developed can be used as a high throughput screening method for drugs and endocrine disrupting chems.

L3 ANSWER 2 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2004:292071 Document No. 140:320040 36Fusion proteins comprising CD1d complex, α 2 microglobulin and antibody or fragment for targeting therapy of tumor, autoimmune disease, inflammation and infection. Robert, Bruno; Donda, Alena; Cesson, Valerie; Mach, Jean-Pierre; Zauderer, Maurice (Vaccinex, Inc., USA). PCT Int. Appl. WO 2004029206 A2 20040408, 152 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US30238 20030926. PRIORITY: EP 2002-405838 20020927.

AB The invention is directed to a compound comprising one or more CD1d complexes in association with an antibody specific for a cell surface marker. The CD1d complexes comprise a CD1d, a ss2-microglobulin mol., and may further comprise an antigen bound to the CD1d binding groove. The invention is further directed to methods of inhibiting or stimulating an immune response with the CD1d-antibody compds., in particular anti-tumor and autoimmunity responses.

L3 ANSWER 3 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2004:220226 Document No. 140:269518 Chimeric IgE polypeptides and aluminum adjuvants as vaccines for treating allergies. Hellman, Lars T.; Persson, Stephen; Gansson, Asa (Resistentia Pharmaceuticals Ab, Swed.). PCT Int. Appl. WO 2004022094 A1 20040318, 102 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-IB3075 20030602. PRIORITY: US 2002-PV408648 20020905.

AB The invention provides methods and materials related to vaccines against self IgE polypeptides. Compns. are described comprising a chimeric self IgE polypeptide and an adjuvant, which is either an aluminum compound or MN51, i.e. Montanide Incomplete Adjuvant (ISA) 51, which is mannide oleate in mineral oil solution Administration of said composition should elicit an anti-self response and thereby reduce the level of detectable free IgE.

L3 ANSWER 4 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2004:162573 Document No. 140:223257 Methods for treating RAGE (receptor for advanced glycation end products)-associated disorders using fusion products of RAGE-LBE domain with Ig element, and genetic constructs encoding same. Pittman, Debra D.; Clancy, Brian; Larsen, Glenn; Trepicchio, William L.; Brennan, Fionula Mary; Feldmann, Marc; Foxwell, Brian John Maurice; Feldman, Jeffrey L. (Wyeth, John, and Brother Ltd., USA). PCT Int. Appl. WO 2004016229 A2 20040226, 100 pp. DESIGNATED

STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US25996 20030818. PRIORITY: US 2002-PV404205 20020816.

AB Fusion proteins comprising a Receptor for Advanced Glycation End Products Ligand Binding Element (RAGE-LBE) and an Ig element are disclosed. A RAGE-LBE may be any extracellular portion of a RAGE protein that retains the ability to bind to a RAGE ligand. Also disclosed are fusion proteins comprising a RAGE-LBE and a dimerization domain. Also disclosed are nucleic acids encoding such fusion proteins and methods for using disclosed nucleic acids and proteins to, for example, treat RAGE-related disorders, such as arthritis. Addnl. compns. and methods are also disclosed.

L3 ANSWER 5 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2004:60344 Document No. 140:124368 Immunoconjugates of modified native human FVIIa with high affinity to tissue factor and therapeutic uses thereof. Bjorn, Soren E.; Nicolaisen, Else Marie; Steenstrup, Thomas D. (Novo Nordisk A/S, Den.). PCT Int. Appl. WO 2004006962 A2 20040122, 61 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-DK481 20030709. PRIORITY: DK 2002-1099 20020712.

AB The present invention relates to novel compds. which bind to and inhibit the activity of tissue factor (TF) and mediate a cytolytic immune response. The invention relates to a immunoconjugates of native humane FVIIa or procoagulant variants that have been chemical inactivated in vitro by covalent active site inhibitors e.g. chloromethyl ketones. The conjugate has very high affinity for TF due to the increased affinity of the chemical inactivated binding domain as compared to the binding of native FVII. The high affinity will provide a more efficacious and safe treatment of a patient in need thereof. The invention also relates to pharmaceutical compns. comprising the novel compds. as well as their use in the treatment of or prophylaxis of diseases or disorders related to pathophysiol. TF activity including cancer, inflammation, atherosclerosis and ischemia/reperfusion.

L3 ANSWER 6 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2004:2625 Document No. 140:75936 Vaccines comprising membrane IgE epitope and non-IgE helper T cell epitope for suppressing IgE-mediated allergic disease. Levinson, Arnold I.; Calarota, Sandra; Weiner, David B.; Otero, Miguel (The Trustees of the University of Pennsylvania, USA). PCT Int. Appl. WO 2004000217 A2 20031231, 37 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US19383 20030620. PRIORITY: US 2002-PV390304 20020620.

AB Nucleic acid mols. that encode a protein comprising at least one epitope of membrane IgE free of epitopes present on the serum IgE, including

proteins that further comprise non-IgE T cell helper epitope are disclosed. Vaccines, vectors and host cells that comprise such nucleic acid mols. are disclosed. Isolated proteins, including haptenized proteins, comprising at least one epitope of membrane IgE free of epitopes present on the serum IgE, including proteins that further comprise non-IgE T cell helper epitope are disclosed. Vaccines that comprise and methods of making such proteins and antibodies that specifically bind to such proteins are disclosed. Killed or inactivated cells or particles, including haptenized killed or inactivated cells or particles, that comprise a protein comprising at least one epitope of membrane IgE free of epitopes present on the serum IgE, including proteins that further comprise non-IgE T cell helper epitope are disclosed. Vaccines that comprise such killed or inactivated cells or particles are disclosed. Methods of treating and preventing IgE mediated allergic disease or condition are disclosed.

L3 ANSWER 7 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:777622 Document No. 139:275730 Antibody fusion proteins: effective adjuvants of protein vaccination. Penichet, Manuel L.; Morrison, Sherie L.; Peng, Lisan; Dela Cruz, Jay (The Regents of the University of California, USA). PCT Int. Appl. WO 2003080106 A1 20031002, 90 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US9136 20030321. PRIORITY: US 2002-PV366917 20020321; US 2002-118473 20020405.

AB The authors disclose antibody-immunostimulant fusion proteins as adjuvants of antigenic protein vaccinations that elicit humoral and/or cellular immune responses in vaccinated subjects. In one example, humoral and cellular immune responses against HER2 were induced by an anti-HER2 IgG3 chimeric antibody expressing the variable domains of Herceptin.

L3 ANSWER 8 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:737855 Document No. 139:255349 Granulocyte colony-stimulating factor fusion proteins with extended serum-lives for stimulation of neutrophil formation. Beals, John Michael; Kuchibhotla, Uma (Eli Lilly and Company, USA). PCT Int. Appl. WO 2003076567 A2 20030918, 126 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-US3120 20030221. PRIORITY: US 2002-PV361948 20020305.

AB Hyperglycosylated fusion proteins of granulocyte colony-stimulating factor (G-CSF) and proteins such as albumin and the Fc portion of an Ig which extend the in vivo half-life of the protein compared to native G-CSF are manufactured by expression of the corresponding gene in an animal cell host. These fusion proteins are suited for the treatment of conditions treatable by stimulation of circulating neutrophils, such as after chemotherapy regimens or in chronic congenital aneupenia. Construction of genes for a number of variants of G-CSF with addnl. glycosylation sites is described.

L3 ANSWER 9 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:874770 Document No. 139:363615 Neutralization of immune suppressive factors for immunotherapy of cancer. Lattime, Edmund C.; Monken, Claude E. (University of Medicine & Dentistry of New Jersey, USA). U.S. Pat.

Appl. Publ. US 2003206886 A1 20031106, 25 pp. (English). CODEN: USXXCO.
APPLICATION: US 2002-138783 20020503.

AB The invention provides vectors comprising nucleic acid mols. that encode polypeptides capable of binding immune suppressive factors for the immunotherapy of cancer. In one example, an immunoadhesin of the interleukin-10 receptor is used to neutralize free ligand.

L3 ANSWER 10 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:492421 Document No. 139:67785 Antigen-binding domain-immunoglobulin fusion proteins for treating malignancies and B cell disorders. Ledbetter, Jeffrey A.; Hayden-Ledbetter, Martha S.; Thompson, Peter A. (Genecraft, Inc., USA). U.S. Pat. Appl. Publ. US 2003118592 A1 20030626, 157 pp., Cont.-in-part of U.S. Ser. No. 53,530. (English). CODEN: USXXCO. APPLICATION: US 2002-207655 20020725. PRIORITY: US 2001-PV367358 20010117; US 2002-53530 20020117; US 2002-PV385691 20020603.

AB The invention relates to novel binding domain-Ig fusion proteins that feature a binding domain for a cognate structure such as an antigen, a counterreceptor or the like, a hinge region polypeptide having either zero or one cysteine residues, and Ig CH2 and CH3 domains, and that are capable of antibody-dependent cell-mediated cytotoxicity (ADCC) and/or complement-dependent cytotoxicity (CDC) while occurring predominantly as monomeric polypeptides. The Ig is Ig heavy or light chain variable region of human Ig, and the antigen is CTLA-4, CD154, CD3, CD19, CD20, CD28, CD37, CD40, CD152, and L6. The fusion proteins can be recombinantly produced at high expression levels. Also provided are related compns. and methods, including immunotherapeutic applications on malignant condition or B cell disorder such as rheumatoid arthritis, myasthenia gravis, Graves' disease, type I diabetes mellitus, multiple sclerosis, and autoimmune disease..

L3 ANSWER 11 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:241912 Document No. 138:265639 Inhibiting the degranulation in mastocytes using a hybrid protein comprising a receptor-binding protein fused to a protease cleaving a protein of the secretion process. Bigalke, Hans; Frevert, Jurgen (Biotecon Gesellschaft Fur Biotechnologische Entwicklung Und Consulting Mbh, Germany). U.S. Pat. Appl. Publ. US 2003059912 A1 20030327, 7 pp., Cont.-in-part of U.S. Ser. No. 700,540. (English). CODEN: USXXCO. APPLICATION: US 2002-64903 20020827. PRIORITY: DE 1998-19821285 19980513; WO 1999-EP3272 19990512; US 2001-700540 20010119.

AB A hybrid protein is provided containing a protein that binds to a receptor of mastocytes and basophils and is endocytosed by them. The protein can be IgE, IgE fragment, IgE Fc fragment, antibody against the IgE receptor of mastocytes and basophils, a fragment of the antibody against the IgE receptor of mastocytes and basophils, an antibody against mastocyte-specific potassium channel, or mast cell degranulating peptide. The hybrid protein also contains a protease which cleaves proteins of the secretion process of the mastocytes and basophils so as to inhibit the secretion process without killing the mastocytes and basophils. The protease can be the light chain of Clostridium botulinum toxin or its proteolytic fragments containing a His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His sequence, the light chain of the tetanus toxin or proteolytically active fragment of the light chain containing His-Asp-Leu-Ile-His-Val-Leu-His, or an IgA protease of Neisseria gonorrhoeae and its proteolytic domain. Thus, a hybrid protein comprising IgE fused to the light chain of either Clostridium botulinum toxin or tetanus toxin prevents allergic shock caused by dying mastocytes.

L3 ANSWER 12 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:365189 Document No. 139:148170 Construction of an sIgE:FLAG-mIgE:GFP Reporter Mouse Strain. Achatz-Straussberger, Gertrude; Geisberger, Roland; Oberndorfer, Iris; Infuehr, Daniela; Luger, Elke; Fallon, Padraic; Lamers, Marinus; Achatz, Gernot (Department of Genetics and General Biology, Salzburg, Austria). International Archives of Allergy and Immunology, 130(4), 280-287 (English) 2003. CODEN: IAAIEG. ISSN:

1018-2438. Publisher: S. Karger AG.

AB Like all other Igs, IgE can be secreted into the blood or expressed as a membrane receptor on the surface of B lymphocytes. Secreted Igs trace the antigen and contribute to its destruction. Membrane Igs accompany the B cell along its differentiation pathway, regulating processes like the induction and maintenance of immunol. memory and differentiation of plasma cells. The regulation of the expression of IgE is very complex. A lot of pos. and neg. regulators influence the synthesis of IgE. In previous publications, the authors were able to show that the membrane IgE (mIgE) antigen receptor itself controls the quantity and quality of serum IgE produced. However, the knowledge about the regulatory function of the antigen receptor on these processes is at best limited. In the present paper, the authors present the construction of a reporter mouse strain, which will help the authors to follow an mIgE-bearing B cell during the immune response more precisely.

L3 ANSWER 13 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2002:716299 Document No. 137:246549 Immunoglobulin fusion proteins that target low-affinity Fcγ receptors. Arnason, Barry G. W.; Jensen, Mark A.; White, David M. (University of Chicago, USA). PCT Int. Appl. WO 2002072608 A2 20020919, 139 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2002-US7365 20020311. PRIORITY: US 2001-PV274392 20010309.

AB The present invention concerns a family of nucleic acids, polypeptides and cloning vectors which direct expression of fusion proteins that can mimic aggregated IgG (AIG) and immune complex function with respect to their interactions with FcγR and which allow for the inclusion and targeting of a second protein domain to cells expressing FcγR. This was accomplished by expressing multiple linear copies of the hinge and CH2 domains (HCH2) of human IgG1 fused to the Fc region of human IgG1. Convenient restriction sites allow for the facile introduction of addnl. N-terminal domains. In one example, the extracellular domain of human CD8α was fused with 0-4 HCH2 segments and the Fc region of IgG1. The fusion protein was shown to stimulate proliferation of interleukin-2-primed natural killer cells.

L3 ANSWER 14 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2002:408803 Document No. 137:1503 Fusion protein of immunoglobulin heavy chain constant region and β-amyloid fragment as therapeutic agent for Alzheimer's disease. Gefter, Malcolm L.; Israel, David I.; Joyal, John L.; Gosselin, Michael (Praecis Pharmaceuticals Inc., USA). PCT Int. Appl. WO 2002042462 A2 20020530, 79 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2001-US44581 20011127. PRIORITY: US 2000-PV253302 20001127; US 2000-PV250198 20001129; US 2000-PV257186 20001220.

AB The present invention provides therapeutic agents and methods of use thereof for treating an amyloidogenic disease, e.g., Alzheimer's disease. The therapeutic agents of the invention include compds. comprising the formula 1-L-P, wherein I is an Ig heavy chain constant region or fragment thereof (e.g., comprising the Fc region); L is a linker group or a direct bond; and P is a peptide capable of binding an amyloidogenic protein. It

is believed that the P portion of the compds. of the invention will serve to bind an amyloidogenic protein, e.g., an amyloidogenic protein within an amyloid plaque, and the I portion of the compds. of the invention will serve to direct microglia to the amyloidogenic protein, which microglia may then internalize and degrade the amyloidogenic protein and the amyloid plaque. COS cells were transfected with DNA encoding various segments of β -amyloid flanked by the mouse IgG1 Fc region. COS cells expressing the Fc Region of mouse IgG1 fused to amino acid residues 1-40, 1-42, 10-25, 16-30, 17-21, or 17-21-(A21L) of β -amyloid with or without an N-terminal triple glycine cap were resolved by SDS-PAGE in the absence of a reducing agent and examined by Western blot anal. The ability of a compound of the invention to modulate (e.g., inhibit or promote) the aggregation of natural β -AP when combined with the natural β -AP was examined using the Fibril binding assay. The results from this experiment (set forth in Figure 9), demonstrate that the compds. tested [e.g., PPI-1019, PPI-1621 and three different preps. of A β (16-30)-Fc] are effective inhibitors of A β aggregation. The ability of A β (16-30)-Fc to clear amyloid plaques in a mouse model of Alzheimer's disease was assessed. The fusion protein was administered to a mouse transgenic for both the Swedish mutation of amyloid precursor protein and presenilin M146L by direct infusion into the cerebral cortex in one hemisphere. As indicated in Figure 10, the plaque burden at the site of infusion was significantly decreased compared to the controlled hemisphere.

L3 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2003:468095 Document No. 139:357756 Chimeric proteins: A novel approach for eliminating specific cell populations for targeted human therapy. Ben-Yehudah, Ahmi; Belostotsky, Ruth; Ageilan, Rami; Azar, Yehudith; Steinberger, Ida; Fishman, Ala; Nechushtan, Amotz; Yarkoni, Shai; Lorberboum-Galski, Haya (Department of Cellular Biochemistry and Human Genetics, Hebrew University-Hadassah Medical School, Jerusalem, 91120, Israel). Cellular and Molecular Mechanisms of Toxin Action, Volume 4, 148-167. Editor(s): Lazarovici, Philip. Taylor & Francis Ltd.: London, UK. (English) 2002. CODEN: 64JPAO.

AB A review. One of the most widely used toxins in chimeric proteins is the bacterial toxin *Pseudomonas* exotoxin (PE) produced by the bacterium *Pseudomonas aeruginosa*. Various chimeric proteins were constructed using two modified forms of the PE toxin: (a) in which Domain I is deleted, generating the PE40 truncated form of PE, (b) by introducing mutations into the binding domain (Domain I) of PE (at amino acid positions 57, 246, 247, 249, all substituted by Glu) to generate the PE664GOU mutated form of PE. The authors designed a number of chimeric proteins for the cure of unrelated disorders: autoimmune diseases, allergy and cancer. For each of these diseases the authors constructed chimeric proteins carrying a specific targeting moiety: Interleukin-2 (IL2) for eliminating activated T cells involved in many human diseases, myelin basic protein (MBP) for therapy of multiple sclerosis (MS), Fc ϵ for use in the treatment of asthma and other allergic disorders and gonadotropin releasing hormone (GnRH) for targeting adenocarcinomas.

L3 ANSWER 16 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

2001:50815 Document No. 134:130249 Fc ϵ receptor fusions with chemi- or bioluminescence-inducing proteins and their uses in IgE detection. Weber, Eric R.; Wood, Keith V.; Hall, Mary P. (Heska Corporation, USA; Promega Corporation). PCT Int. Appl. WO 2001004310 A1 20010118, 104 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-US19070 20000713. PRIORITY: US 1999-PV143612 19990713; US 2000-PV186412 20000302.

AB The present invention relates to chimeric genes for Fc ϵ receptor

fused to bioluminescence- or chemiluminescence-inducing proteins, fusion proteins encoded by such nucleic acid mols., and methods of using such proteins and nucleic acid mols. for the detection of IgE and for identifying compds. capable of inhibiting Fcε receptor activity. Thus, chimeric genes encoding human Fcε receptor extracellular domain fused to luciferase or alkaline phosphatase were constructed and expressed in Escherichia coli. These fusion proteins were used in detection of anti-flea saliva antigen or anti-Dermatophagoides pteronyssinus antigen IgE in allergy patients' sera.

L3 ANSWER 17 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

1998:178127 Document No. 128:214189 Recombinant protein expression and secretion by mammalian host cell using mammalian signal sequence and immunoglobulin Fc region fusion with target protein (immunofusins). Lo, Kin-ming; Sudo, Yukio; Gillies, Stephen D. (Fuji ImmunoPharmaceuticals Corp., USA). U.S. US 5726044 A 19980310, 18 pp., Cont.-in-part of U.S. 5,541,087. (English). CODEN: USXXAM. APPLICATION: US 1995-528122 19950914. PRIORITY: US 1994-305700 19940914.

AB Disclosed are DNAs produced by recombinant techniques for inducing the expression and subsequent secretion of a target protein. The DNAs encode, in their 5' to 3' direction, a secretion cassette, including a signal sequence and an Ig Fc region, and a target protein. The DNAs can be transfected into a host cell for the expression, production and subsequent secretion of the target protein as a fusion protein. The secreted protein can be collected from the extracellular space, and further purified as desired. The secreted fusion protein addnl. can be proteolytically cleaved to release the target protein from the secretion cassette. An exemplary secretion cassette comprises the signal sequence of an Ig light chain of the 14.18 antibody modified for ease of cloning, the Fc region of human Fcγ1 genomic DNA (including the genomic configuration of the hinge, CH2 and CH3 regions), a proteolytic cleavage site-encoding sequence, and DNA sequences encoding target proteins such as CD26, interleukin-2, HIV Tat or Rev proteins, OSF-2 secretory protein involved in ossification, etc.

L3 ANSWER 18 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN

1996:350365 Document No. 125:27685 Recombinant protein expression and secretion by mammalian host cell using mammalian signal sequence and immunoglobulin Fc region fusion with target protein. Lo, Kin-Ming; Sudo, Yukio; Gillies, Stephen D. (Fuji Immunopharmaceuticals Corporation, USA). PCT Int. Appl. WO 9608570 A1 19960321, 51 pp. DESIGNATED STATES: W: AU, CA, JP; RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1995-US11720 19950914. PRIORITY: US 1994-305700 19940914.

AB Disclosed are DNAs produced by recombinant techniques for inducing the expression and subsequent secretion of a target protein. The DNAs encode, in their 5' to 3' direction, a secretion cassette, including a signal sequence and an Ig Fc region, and a target protein. The DNAs can be transfected into a host cell for the expression, production and subsequent secretion of the target protein as a fusion protein. The secreted protein can be collected from the extracellular space, and further purified as desired. The secreted fusion protein addnl. can be proteolytically cleaved to release the target protein from the secretion cassette.

L3 ANSWER 19 OF 21 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

1994:46608 Document No.: PREV199497059608. High level expression and characterization of a recombinant CD4-IgE-fusion protein. Kufer, P.; Krauss, S.; Person, S.; Federle, C.; Rieber, E. P.; Riethmueller, G.. Inst. Immunol., Muenchen, Germany. Immunobiology, (1993) Vol. 189; No. 1-2, pp. 228. Meeting Info.: 24th Meeting of the Society for Immunology. Leipzig, Germany. September 30-October 2, 1993. CODEN: IMMND4. ISSN: 0171-2985. Language: English.

L3 ANSWER 20 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
 1990:49998 Document No. 112:49998 Cloning and expression of cDNA for soluble CD4 derivatives and fusion proteins. Capon, Daniel J.; Gregory, Timothy J. (Genentech, Inc., USA). Eur. Pat. Appl. EP 314317 A1 19890503, 36 pp. DESIGNATED STATES: R: ES, GR. (English). CODEN: EPXXDW. APPLICATION: EP 1988-309194 19881003. PRIORITY: US 1987-104329 19871002; US 1988-250785 19880928.

AB Water-soluble derivs. of the CD4 antigen and water-soluble fusions of CD4 with Ig polypeptides that are potentially useful as therapeutic agents are described. A series of CD4-herpesvirus glycoprotein D fusion proteins were prepared and their interaction with human immunodeficiency virus (HIV) gp120 studies. Binding consts. for the interaction were .apprx.10⁻⁹M. The soluble fusion proteins also greatly reduced the infection of culture cells by HIV.

L3 ANSWER 21 OF 21 CAPLUS COPYRIGHT 2004 ACS on STN
 1986:220129 Document No. 104:220129 Production of immunogleukin EC22 by transformed Escherichia coli. Senoo, Masaharu; Onda, Haruo; Igarashi, Koichi (Takeda Chemical Industries, Ltd., Japan). PCT Int. Appl. WO 8504673 A1 19851024, 36 pp. DESIGNATED STATES: W: MC. (Japanese). CODEN: PIXXD2. APPLICATION: WO 1984-JP181 19840410.

AB A recombinant DNA is produced by ligating a DNA fragment that encodes an antibody recognition site (e.g., IgE) with a DNA fragment that encodes a human interleukin 2 (HIL-2). Escherichia coli Transformed with a plasmid containing the recombinant DNA produces a polypeptide consisting of the human IgE and human IL-2 called immunogleukin EC22 (IGL-EC22). Thus, plasmid pGEL1028 was constructed by ligating an 8-kilobase (kb) DNA fragment containing the gene encoding the C2 region of the human IgE heavy chain, which was isolated from the E. coli plasmid pGETtrp818-C, with a 500-kb DNA fragment containing the gene for HIL-2 from E. coli plasmid pTF1. E. coli Were transformed with plasmid IGL-EC22. The nucleotide sequence, its encoded amino acid sequence, and a restriction map of the recombinant DNA are given. The product immunogleukin EC22 is a useful reagent for purification of human interleukin 2 antibody.

=> s "CH3" and "CH4" fusion
 L4 0 "CH3" AND "CH4" FUSION

=> s IgE Fc fusion
 L5 6 IGE FC FUSION

=> dup remove l5
 PROCESSING COMPLETED FOR L5
 L6 2 DUP REMOVE L5 (4 DUPLICATES REMOVED)

=> d l6 1-2 cbib abs

L6 ANSWER 1 OF 2 MEDLINE on STN DUPLICATE 1
 2004433011. PubMed ID: 15316510. Inhibition of allergen-specific IgE reactivity by a human Ig Fcgamma-Fcepsilon bifunctional fusion protein. Zhang Ke; Kopley Christopher L; Terada Tetsuya; Zhu Daocheng; Perez Hector; Saxon Andrew. (Hart and Louis Lyon Laboratory, Division of Clinical Immunology and Allergy, Department of Medicine, University of California Los Angeles School of Medicine, CA 90095-1680, USA.) Journal of allergy and clinical immunology, (2004 Aug) 114 (2) 321-7. Journal code: 1275002. ISSN: 0091-6749. Pub. country: United States. Language: English.

AB BACKGROUND: Coaggregating FcepsilonRI with FcgammaRII receptors holds great potential for treatment of IgE-mediated disease by inhibiting FcepsilonRI signaling. We have previously shown that an Fcgamma-Fcepsilon fusion protein, human IgG-IgE Fc fusion protein (GE2), could inhibit FcepsilonRI-mediated mediator releases in vitro and in vivo. OBJECTIVE: We sought to test whether GE2 was capable of blocking mediator release from FcepsilonRI cells sensitized with IgE in

vivo or in vitro before exposure to GE2, a critical feature for GE2 to be clinically applicable. METHODS: GE2 was tested for its ability to inhibit Fel d 1-induced mediator release from human blood basophils from subjects with cat allergy, human lung-derived mast cells, human FcepsilonRIalpha transgenic mice sensitized with human cat allergic serum, and rhesus monkeys naturally allergic to the dust mite Dermatophagoides farinae. RESULTS: Basophils from subjects with cat allergy and lung mast cells degranulate when challenged with Fel d 1 and anti-IgE, respectively. GE2 itself did not induce mediator release but strongly blocked this Fel d 1- and anti-IgE-driven mediator release. GE2 was able to block Fel d 1-driven passive cutaneous anaphylaxis at skin sites sensitized with human serum from subjects with cat allergy in human FcepsilonRIalpha transgenic mice, but by itself, GE2 did not induce a passive cutaneous anaphylaxis reaction. Finally, GE2 markedly inhibited skin test reactivity to D farinae in monkeys naturally allergic to this allergen, with complete inhibition being observed at 125 ng. CONCLUSION: GE2 is able to successfully compete for FcepsilonRs and FcgammaRs on cells presensitized in vitro and in vivo and lead to inhibition of IgE-mediated reactivity through coaggregation of FcepsilonRI with FcgammaRII.

L6 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN

1988:588798 Document No. 109:188798 Immunoaffinity column purification of recombinant IgE Fc fragment fusion proteins for use as an antiallergy medicine. Ikeyama, Shuichi; Nishimura, Osamu (Takeda Chemical Industries, Ltd., Japan). Eur. Pat. Appl. EP 269455 A2 19880601, 19 pp. DESIGNATED STATES: R: AT, BE, CH, DE, ES, FR, GB, GR, IT, LI, LU, NL, SE. (English). CODEN: EPXXDW. APPLICATION: EP 1987-310475 19871127. PRIORITY: JP 1986-281871 19861128; JP 1987-232295 19870918.

AB Purification of recombinant IgE Fc fragment-interleukin-2 (IL-2) signal peptide fusion proteins using immunoaffinity chromatog. Mouse L cells transformed with pTB543, encoding a fusion protein comprising the IL-2 leader peptide, the 1st 11 N-terminal amino acids of IL-2, a small linker peptide, and the Fc portion of human IgE, were cultured. The fusion protein was purified from the medium using (NH4)2SO4 precipitation, immunoaffinity chromatog. (with

a monoclonal antibody against IgE), and gel filtration chromatog. (Sephacryl S-200). A pure glycosylated protein was obtained.

=> S hellman l?/au

L7 1235 HELLMAN L?/AU

=> s l7 and "CH3-CH4 fusion"

L8 0 L7 AND "CH3-CH4 FUSION"

=> dup remove l7

PROCESSING COMPLETED FOR L7

L9 572 DUP REMOVE L7 (663 DUPLICATES REMOVED)

=> s l9 and CH3 IgE domain

L10 0 L9 AND CH3 IGE DOMAIN

=> s l9 and vaccine

L11 14 L9 AND VACCINE

=> dup remove l11

PROCESSING COMPLETED FOR L11

L12 14 DUP REMOVE L11 (0 DUPLICATES REMOVED)

=> d l12 1-14 cbib abs

L12 ANSWER 1 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN

2004:490733 Document No. 141:52843 Complement C5 immunogens for treating inflammatory conditions. Hellman, Lars T.; Holmdahl, Rikard (Resistentia Pharmaceuticals AB, Swed.). PCT Int. Appl. WO 2004050111 A2

20040617, 52 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-IB6344 20031201.

PRIORITY: US 2002-PV430278 20021202.

AB The authors disclose polypeptide immunogens, and their isolated nucleic acids, that can be used to induce an antibody response in a mammal against complement C5 or C5a. In one example, a MBP-C5a fusion protein is shown to inhibit joint inflammation development in a collagen-induced arthritis model.

L12 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN

2004:220226 Document No. 140:269518 Chimeric IgE polypeptides and aluminum adjuvants as **vaccines** for treating allergies. **Hellman, Lars T.**; Persson, Stephen; Gansson, Asa (Resistentia Pharmaceuticals Ab, Swed.). PCT Int. Appl. WO 2004022094 A1 20040318, 102 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2003-IB3075 20030602. PRIORITY: US 2002-PV408648 20020905.

AB The invention provides methods and materials related to **vaccines** against self IgE polypeptides. Compns. are described comprising a chimeric self IgE polypeptide and an adjuvant, which is either an aluminum compound or MN51, i.e. Montanide Incomplete Adjuvant (ISA) 51, which is mannide oleate in mineral oil solution Administration of said composition should elicit an anti-self response and thereby reduce the level of detectable free IgE.

L12 ANSWER 3 OF 14 MEDLINE on STN

2004345109. PubMed ID: 15246623. Identification of adjuvants that enhance the therapeutic antibody response to host IgE. Johansson Jeannette; Ledin Anna; Vernersson Molly; Lovgren-Bengtsson Karin; **Hellman Lars**. (Department of Cell and Molecular Biology, Biomedical Center, Box 596, Uppsala University, S-751 24, Sweden.) Vaccine, (2004 Jul 29) 22 (21-22) 2873-80. Journal code: 8406899. ISSN: 0264-410X. Pub. country: Netherlands. Language: English.

AB In the development of a novel **vaccine** against atopic allergies, we have screened for adjuvants that enhance the therapeutic antibody response against self immunoglobulin E (IgE). The response against self IgE is induced by administration of a **vaccine** antigen, which contains both self and non-self IgE regions, together with an adjuvant. We evaluated five commonly used adjuvants; Freund's, aluminium hydroxide, ISCOMs, Montanide ISA 51 and Montanide ISA 720, and found that the mineral oil-based adjuvants; Montanide ISA 51 and Freund's induced at least 5-10-fold higher anti-self IgE titers than any of the other candidates. However, with one exception, Alum, the immune responses against the carrier, i.e. the non-self regions, were similar for all adjuvants, indicating that the ability to induce responses against self and non-self antigens differ among adjuvants. The responses against non-self IgE were more than 50-fold higher than antibody responses against self IgE in both the Freund's and Montanide 51-administered animals, indicating that the response against self molecules is markedly inhibited by tolerance-inducing mechanisms. Co-administration of Montanide ISA 51 with

immuno-stimulatory substances from bacteria; muramyl dipeptide (MDP), monophosphoryl-lipid A (MPL) or a formyl-methionine-containing tripeptide (fMLP), did not elevate the anti-self IgE response. Hence, adjuvants based on pure mineral oil without additional immuno-stimulatory substances appear to be the best adjuvant candidates in therapeutic vaccines aimed at regulating the in vivo levels of self-proteins.

L12 ANSWER 4 OF 14 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

2003:359832 Document No.: PREV200300359832. Generation of a therapeutic anti-IgE response in a primate model through vaccination. Persson, S. [Reprint Author]; Lundgren, M. [Reprint Author]; Hellman, L. [Reprint Author]. Resistencia Pharmaceuticals AB, Uppsala, Sweden. Journal of Allergy and Clinical Immunology, (February 2003) Vol. 111, No. 2 Abstract Supplement, pp. S285. print.
Meeting Info.: AAAAI 60th Anniversary Meeting. Denver, CO, USA. March 07-12, 2003. American Academy of Allergy, Asthma and Immunology.
CODEN: JACIBY. ISSN: 0091-6749. Language: English.

L12 ANSWER 5 OF 14 MEDLINE on STN

2002300838. PubMed ID: 11967231. Generation of therapeutic antibody responses against IgE through vaccination. Vernerersson Molly; Ledin Anna; Johansson Jeannette; Hellman Lars. (Department of Cell and Molecular Biology, Biomedical Center, University of Uppsala, S-751 24 Uppsala, Sweden.) FASEB journal : official publication of the Federation of American Societies for Experimental Biology, (2002 Jun) 16 (8) 875-7. Journal code: 8804484. ISSN: 1530-6860. Pub. country: United States. Language: English.

AB IgE is the central mediator in atopic allergies such as hay fever, eczema, and asthma; therefore, it is a prime target in the development of allergen-independent preventive treatments. We describe an active immunization strategy that has the potential to reduce IgE to a clinically significant extent. The active vaccine component is a chimeric IgE molecule, Cepsilon2-Cepsilon3-Cepsilon4. The receptor-binding target domain, Cepsilon3, is derived from the recipient species, whereas the flanking domains, Cepsilon2 and Cepsilon4, are derived from an evolutionarily distant mammal. The flanking domains have dual functions, acting both as structural support for the Cepsilon3 domain and to break T cell tolerance by providing foreign T cell epitopes. The efficacy of the vaccine was studied in an ovalbumin-sensitized rat model. Vaccination resulted in antibody responses against IgE in all rats and in a substantial reduction in serum IgE levels in three out of four strains. The skin reactivity upon allergen challenge was significantly reduced in vaccinated animals. The vaccine appears to be safe to use as an antigen. No cross-linking activity was observed in sera of vaccinated animals, and the response to vaccination was reversible with time. Our results suggest that active immunization against IgE has the potential to become a therapeutic method for humans.

L12 ANSWER 6 OF 14 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

2002:395291 The Genuine Article (R) Number: 549CA. Generation of therapeutic antibody responses against IgE through vaccination. Vernerersson M; Ledin A; Johansson J; Hellman L (Reprint). Uppsala Univ, Ctr Biomed, Dept Cell & Mol Biol, Box 596, S-75124 Uppsala, Sweden (Reprint); Uppsala Univ, Ctr Biomed, Dept Cell & Mol Biol, S-75124 Uppsala, Sweden; Resistencia Pharmaceut AB, S-75323 Uppsala, Sweden. FASEB JOURNAL (APR 2002) Vol. 16, No. 6, pp. U104-U124. Publisher: FEDERATION AMER SOC EXP BIOL. 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA. ISSN: 0892-6638. Pub. country: Sweden. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB IgE is the central mediator in atopic allergies such as hay fever, eczema, and asthma; therefore, it is a prime target in the development of allergen-independent preventive treatments. We describe an active immunization strategy that has the potential to reduce IgE to a clinically

significant extent. The active **vaccine** component is a chimeric IgE molecule, Cepsilon2-Cepsilon3-Cepsilon4. The receptor-binding target domain, Cepsilon3, is derived from the recipient species, whereas the flanking domains, Cepsilon2 and Cepsilon4, are derived from an evolutionarily distant mammal. The flanking domains have dual functions, acting both as structural support for the Cepsilon3 domain and to break T cell tolerance by providing foreign T cell epitopes. The efficacy of the **vaccine** was studied in an ovalbumin-sensitized rat model. Vaccination resulted in antibody responses against IgE in all rats and in a substantial reduction in serum IgE levels in three out of four strains. The skin reactivity upon allergen challenge was significantly reduced in vaccinated animals. The **vaccine** appears to be safe to use as an antigen. No cross-linking activity was observed in sera of vaccinated animals, and the response to vaccination was reversible with time. Our results suggest that active immunization against IgE has the potential to become a therapeutic method for humans.

L12 ANSWER 7 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN

2000:314492 Document No. 132:346610 Enhanced **vaccines**.

Hellman, Lars T. (Resistentia Pharmaceuticals AB, Swed.). PCT Int. Appl. WO 2000025722 A2 20000511, 50 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-SE1896 19991021. PRIORITY: US 1998-PV106652 19981102; US 1999-401636 19990922.

AB The invention relates to methods and materials involved in the treatment and prevention of various diseases such as infections and IgE-related diseases. Specifically, the invention relates to methods and materials that can be used to vaccinate a mammal against specific self or non-self antigens. For example, the methods and materials described herein can be used to reduce the effects of IgE antibodies within a mammal by reducing the amount of total and receptor bound IgE antibodies in the mammal. In addition, the invention provides **vaccine** conjugates, immunogenic polypeptides, nucleic acid mols. that encode immunogenic polypeptides, host cells containing the nucleic acid mols. that encode immunogenic polypeptides, and methods for making **vaccine** conjugates and immunogenic polypeptides as well as nucleic acid mols. that encode immunogenic polypeptides. Further, the invention provides an IgE **vaccine** that induces an anti-self IgE response in a mammal.

L12 ANSWER 8 OF 14 CAPLUS COPYRIGHT 2004 ACS on STN

1999:56260 Document No. 130:266025 **Vaccines** against allergies.

Hellman, L. (Department of Medical Immunology and Microbiology, BMC, Uppsala, S-751 23, Swed.). Handbook of Experimental Pharmacology, 133 (Vaccines), 499-526 (English) 1999. CODEN: HEPHD2. ISSN: 0171-2004. Publisher: Springer-Verlag.

AB A review with 135 refs. A detailed description of allergic immune response along with a discussion of some of the currently available immunotherapies is presented. Application of modified allergens, oral administration of allergens and allergen exts., peptide **vaccines**, cytokine agonists and antagonists as immunotherapeutic approach is discussed. In addition, use of low mol. weight substances that interfere with the interactions between IgE and its receptors as well as strategies involving the depletion of plasma and mast cell bound IgE by treatment with monoclonal anti-IgE antibodies is also mentioned. Finally, strategies involving induction of strong anti-IgE response by vaccination are outlined.

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2002:81032 Document No.: PREV200200081032. **Vaccine** comprising part of constant region of IgE for treatment of IgE-mediated allergic reactions. **Hellman, L. T.** [Inventor]. Vaderkvarnsgatan 11A, S-753 29 Uppsala, Sweden. Patent Info.: US 5653980 Aug. 5, 1997. Official Gazette of the United States Patent and Trademark Office Patents, (Aug. 5, 1997) Vol. 1201, No. 1, pp. 362. print.
CODEN: OGUPE7. ISSN: 0098-1133. Language: English.

L12 ANSWER 10 OF 14 MEDLINE on STN

97249376. PubMed ID: 9095262. Is vaccination against IgE possible?.

Hellman L. (Department of Medical Immunology and Microbiology, University of Uppsala.) Advances in experimental medicine and biology, (1996) 409 337-42. Ref: 18. Journal code: 0121103. ISSN: 0065-2598. Pub. country: United States. Language: English.

AB A substantial reduction in the levels of both total and antigen specific IgE will most likely result in improved symptom scores in atopic individuals. Based on this assumption we initiated a project to study the possibility of reducing levels of circulating and mast cell bound IgE, by inducing a strong autoimmune antibody response against IgE in the host. Bacterially produced fusion proteins containing constant domains two (CH2) and three (CH3) of rat IgE directly linked to the glutathione-S-transferase (GST) protein from *Schistosoma japonicum* or to the maltose binding protein of *Escherichia coli* were used as the active components of the allergy vaccine. Injection of either of these fusion proteins together with adjuvant led to the induction of a strong autoimmune anti-IgE response in several IgE low or medium responder strains of rats. Vaccination of ovalbumin sensitised Wistar rats with the GST-C2C3 fusion protein resulted in a profound decrease in serum IgE levels and later in a nearly complete block in histamine release from mast cells and basophils upon challenge with either a cross-linking polyclonal anti-IgE antiserum or a specific allergen. This shows that it is possible to reduce IgE levels in an animal to such an extent that it gives a clear clinical effect. Recent studies with an extended panel of rat strains including four IgE high responder strains, indicate that induction of the autoimmune response is dependent on the plasma concentration of IgE before vaccination. A high concentration of IgE has a negative effect on the induction of autoimmunity, most likely by inducing a B-cell tolerance in the host. Vaccinated subjects with very high IgE concentrations thereby responds poorly to the vaccine. Current studies are aimed at overcoming this potential limitation of the vaccination procedure.

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96297255 EMBASE Document No.: 1996297255. Allergy vaccines: A review of developments. **Hellman L.**; Carlsson M.. Department of Medical Immunology, Biomedical Centre, University of Uppsala, S-751 23 Uppsala, Sweden. Clinical Immunotherapeutics 6/2 (130-142) 1996. ISSN: 1172-7039. CODEN: CIMMEA. Pub. Country: New Zealand. Language: English. Summary Language: English.

AB Immunotherapy by vaccination (hyposensitisation) has been used since the beginning of this century for the treatment of atopic diseases. Immunotherapy is still widely used and in the hands of specialists is quite safe. However, the use of crude allergen extracts, doubts about its efficacy for many allergens and the risk of severe adverse effects when not properly administered have raised questions about the place of hyposensitisation as part of modern immunotherapy. The relatively efficient pharmacotherapy of allergic diseases has also reduced the need for traditional high dose immunotherapy. However, progress in the understanding of the basic immune mechanisms of allergy and in the characterisation of dominant allergens has stimulated the development of several novel strategies for immunotherapy. A few of these have the potential of reaching the clinic in the near future. The most promising areas of this rapidly developing field will be covered in this article. The 4 main areas which will be discussed in more detail are: (i) progress in the area of modifications :of allergen extracts or purified recombinant

allergens by allergen cross-linking, monomethoxy-polyethylene glycol coupling or immune complex formation, with the aim of reducing the allergenicity of the antigen or to tolerise or redirect the immune response to a mainly T helper 1 response; (ii) oral administration of allergens or allergen extracts, possibly by using bacteria as live **vaccines**; (iii) treatment with immunodominant peptides from major allergens, with the aim of inducing unresponsiveness in allergen-specific T cells; and (iv) immune intervention directly targeting the IgE molecule, to deplete circulating and mast cell bound IgE, by treatment with monoclonal antibodies or by vaccination against IgE using parts of the IgE molecule covalently coupled to a foreign carrier protein.

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1995:383188 Document No.: PREV199598397488. Analysis of a novel allergy **vaccine** designed for the treatment of IgE-mediated allergies.
Hellman, L.; Carlsson, M.; Aveskogh, M.; Akerlund, R.. Dep. Med. Immunol. Microbiol., Uppsala Univ., Uppsala, Sweden. 9TH INTERNATIONAL CONGRESS OF IMMUNOLOGY. (1995) pp. 437. The 9th International Congress of Immunology. Publisher: 9th International Congress of Immunology, San Francisco, California, USA.
Meeting Info.: Meeting Sponsored by the American Association of Immunologists and the International Union of Immunological Societies. San Francisco, California, USA. July 23-29, 1995.
Language: English.

L12 ANSWER 13 OF 14 MEDLINE on STN

94130960. PubMed ID: 8299691. Profound reduction in allergen sensitivity following treatment with a novel allergy **vaccine**. **Hellman L.** (Department of Immunology, University of Uppsala, Sweden.) European journal of immunology, (1994 Feb) 24 (2) 415-20. Journal code: 1273201. ISSN: 0014-2980. Pub. country: GERMANY: Germany, Federal Republic of. Language: English.

AB A novel approach is described for the treatment of IgE-mediated allergic reactions which is based on the induction of a strong anti-IgE response in the host. Vaccination of ovalbumin-sensitized rats with constant domains two and three of rat IgE coupled to a heterologous carrier protein resulted in a profound decrease in serum levels of IgE, and later in a nearly complete block of histamine release from mast cells and basophils upon challenge with either a cross-linking polyclonal anti-IgE antiserum or a specific allergen.

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1993:219841 Document No. 118:219841 **Vaccine** comprising part of constant region of IgE for treatment of IgE-mediated allergic reactions. **Hellman, Lars T.** (Swed.). PCT Int. Appl. WO 9305810 A1 19930401, 27 pp. DESIGNATED STATES: W: AT, AU, BB, BG, BR, CA, CH, CS, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MG, MN, MW, NL, NO, PL, RO, RU, SD, SE, US; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, DE, DK, ES, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NL, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1992-SE673 19920925. PRIORITY: SE 1991-2808 19910926.

AB A **vaccine** for alleviating the symptoms of or preventing the induction of IgE-mediated allergic reactions in a mammal contains a protein having the entire amino acid sequence of the constant CH2CH3 domains of the ϵ chain of IgE mol. from the mammal species or a structurally stable subunit of said amino acid sequence containing ≥ 12 amino acids, in its original or in a mutated or multimerized form, and optionally containing an adjuvant. The cDNA sequence for CH2CH3 regions of the rat ϵ chain of IgE was cloned and ligated into a com. available vector for the production of a fusion protein (purity of .apprx.50%) in Escherichia coli. Strong immune response was obtained when rats were injected s.c. with 100 μ g of fusion protein in 0.2mL admixt. with an adjuvant.

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L14 ANSWER 1 OF 2 MEDLINE on STN
2002300838. PubMed ID: 11967231. Generation of therapeutic antibody responses against IgE through vaccination. VernerSSon Molly; Ledin Anna; Johansson Jeannette; Hellman Lars. (Department of Cell and Molecular Biology, Biomedical Center, University of Uppsala, S-751 24 Uppsala, Sweden.) FASEB journal : official publication of the Federation of American Societies for Experimental Biology, (2002 Jun) 16 (8) 875-7. Journal code: 8804484. ISSN: 1530-6860. Pub. country: United States. Language: English.
AB IgE is the central mediator in atopic allergies such as hay fever, eczema, and asthma; therefore, it is a prime target in the development of allergen-independent preventive treatments. We describe an active immunization strategy that has the potential to reduce IgE to a clinically significant extent. The active vaccine component is a chimeric IgE molecule, **Cepsilon2-Cepsilon3-Cepsilon4**. The receptor-binding target domain, Cepsilon3, is derived from the recipient species, whereas the flanking domains, Cepsilon2 and Cepsilon4, are derived from an evolutionarily distant mammal. The flanking domains have dual functions, acting both as structural support for the Cepsilon3 domain and to break T cell tolerance by providing foreign T cell epitopes. The efficacy of the vaccine was studied in an ovalbumin-sensitized rat model. Vaccination resulted in antibody responses against IgE in all rats and in a substantial reduction in serum IgE levels in three out of four strains. The skin reactivity upon allergen challenge was significantly reduced in vaccinated animals. The vaccine appears to be safe to use as an antigen. No cross-linking activity was observed in sera of vaccinated animals, and the response to vaccination was reversible with time. Our results suggest that active immunization against IgE has the potential to become a therapeutic method for humans.

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2002:395291 The Genuine Article (R) Number: 549CA. Generation of therapeutic antibody responses against IgE through vaccination. VernerSSon M; Ledin A; Johansson J; Hellman L (Reprint). Uppsala Univ, Ctr Biomed, Dept Cell & Mol Biol, Box 596, S-75124 Uppsala, Sweden (Reprint); Uppsala Univ, Ctr Biomed, Dept Cell & Mol Biol, S-75124 Uppsala, Sweden; Resistencia Pharmaceut AB, S-75323 Uppsala, Sweden. FASEB JOURNAL (APR 2002) Vol. 16, No. 6, pp. U104-U124. Publisher: FEDERATION AMER SOC EXP BIOL. 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA. ISSN: 0892-6638. Pub. country: Sweden. Language: English.

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB IgE is the central mediator in atopic allergies such as hay fever, eczema, and asthma; therefore, it is a prime target in the development of allergen-independent preventive treatments. We describe an active immunization strategy that has the potential to reduce IgE to a clinically significant extent. The active vaccine component is a chimeric IgE molecule, **Cepsilon2-Cepsilon3-Cepsilon4**. The receptor-binding target domain, Cepsilon3, is derived from the recipient species, whereas the flanking domains, Cepsilon2 and Cepsilon4, are derived from an evolutionarily distant mammal. The flanking domains have dual functions, acting both as structural support for the Cepsilon3 domain and to break T cell tolerance by providing foreign T cell epitopes. The efficacy of the vaccine was studied in an ovalbumin-sensitized rat model. Vaccination resulted in antibody responses against IgE in all rats and in a substantial reduction in serum IgE levels in three out of four strains.

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